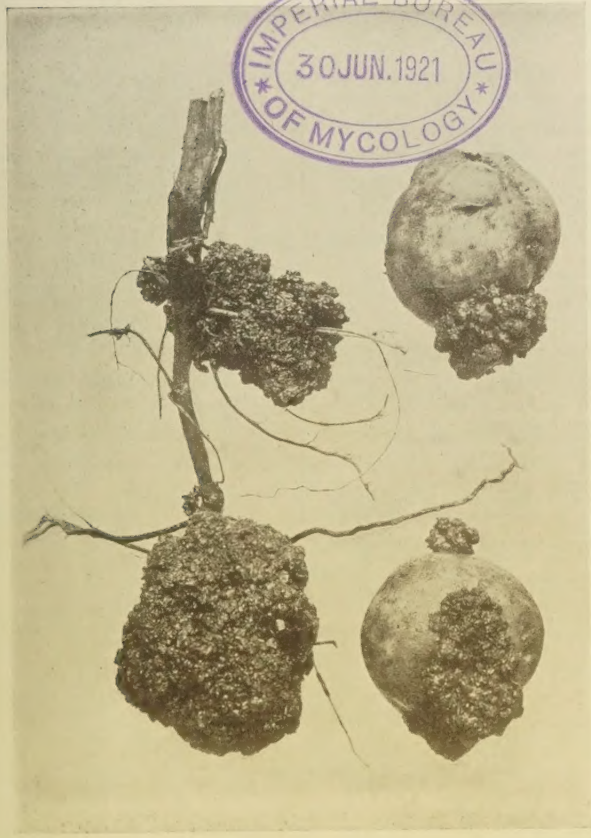
MARCH, 1919 BULLETIN No. 156

THE PENNSYLVANIA STATE COLLEGE

SCHOOL OF AGRICULTURE

Agricultural Experiment Station


IMPERIAL BUREAU
30 JUN. 1921
OF MYCOLOGY

THE POTATO WART DISEASE

A New and Serious Disease Recently Discovered in Pennsylvania

STATE COLLEGE, CENTRE COUNTY, PENNSYLVANIA

THE PENNSYLVANIA STATE COLLEGE
SCHOOL OF AGRICULTURE
AGRICULTURAL EXPERIMENT STATION
Advisory Committee of the Board of Trustees

H. V. WHITE, Chairman, Bloomsburg, Pa.

E. S. BAYARD .....Pittsburgh
VANCE C. McCORMICK ...Harrisburg
C. E. PATTON .....Curwensville
C. J. TYSON .....Flora Dale

EDWIN ERLE SPARKS, President of the College

STAFF

Administration
R. L. Watts, Dean and Director
C. L. Goodling, Superintendent of Farms
Julia C. Gray, Librarian and Editor
William G. Murtorff, Clerk
H. Marilla Williams, Record Clerk
Jean T. Sandstrom, Secretary
Kathryn M. Stanford, Assistant Librarian

Agronomy
F. D. Gardener, Head of Department
E. L. Worthen, Soils
C. F. Noll, Farm Crops
W. H. Darst, Farm Crops
J. W. White, Soil Research
D. S. Fox, Farm Management
R. U. Blasingame, Farm Mechanics
D C. Wimer, Soils
F. J. Holben, Soil Research
J. S. Owens, Farm Crops

Agricultural Education
T. I. Mairs, Head of Department
*W. R. White, Assistant
E. V. Bearer, Assistant

Agricultural Chemistry
Charles W. Stoddart, Head of Department
Margaret B. MacDonald, Food and Dairy Chemistry
*D. E. Haley, Soil and Fertilizer Chemistry
M. W. Lisse, Physiological Chemistry

Animal Husbandry
W. H. Tomhave, Head of Department
M. F. Grimes, Beef Cattle
I. D. Wilson, Veterinarian; Horses
L. S. Kleinschmidt, Poultry
F. L. Bentley, Beef Cattle
C. W. McDonald, Sheep
*Fred Hultz, Sheep

Botany
Frank D. Kern, Head of Department
C. R. Orton, Plant Pathology
J. Ben Hill, Genetics
*J. F. Adams, Plant Pathology
*J. P. Kelly, Genetics
*W. G. Edwards, Botany and Forestry
L. O. Overholts, Mycology
*J. W. Miller, Plant Pathology
R. C. Walton, Plant Pathology (Arendtsville).
W. S. Beach, Plant Pathology (Bustleton)
J. H. Muncie, Plant Pathology (Girard)
E. A. Siegler, Plant Pathology
John Ellis, Gardener

Dairy Husbandry
Fred Rasmussen, Head of Department
E. L. Anthony, Farm Dairying and Milk Production
*Elizabeth B. Meek, Bacteriology
S. I. Bechdel, Milk Production
C. A. Hunter, Bacteriology
*A. L. Beam, Milk Production and Advanced Registry Testing
W. D. Swope, Milk Production
H. C. Yerger, Jr., Dairy Manufacture
Charles Cummings, Farm Dairying and Milk Production
J. F. Olney, Bacteriology
F. P. Knoll, Superintendent of Creamery

Experimental Agricultural Chemistry
William Frear, Vice Director and Head of Department
E. S. Erb, Associate Chemist
Walter Thomas, Associate Chemist
Charles Kern, Laboratory Assistant

Field Laboratories
C. H. Hadley, Economic Entomology (Bustleton)
S. W. Frost, Economic Entomology (Arendtsville)

Forestry
John A. Ferguson, Head of Department
*George R. Green, Wood Technology
Clarence R. Anderson, Forest Management

Horticulture
S. W. Fletcher, Head of Department
C. E. Myers, Plant Breeding
A. W. Cowell, Landscape Art
F. N. Fagan, Pomology
E. I. Wilde, Floriculture
J. R. Bechtel, Vegetable Gardening
J. S. Gardner, Plant Breeding
L. D. Jesseman, Pomology

Rural Life
*W. S. Taylor, Head of Department
R. G. Bressler, Acting Head; Rural Sociology
H. G. Parkinson, Agricultural Education
George Wehrwein, Rural Economics
F. T. Struck, Agricultural Education

*Absent on leave

The bulletins of the Station will be mailed regularly, free of charge, to all residents of the State who request them. Address: Director of Experiment Station, State College, Centre Co., Pa.

Sun Printing & Binding Co., Williamsport, Pa.

THE POTATO WART DISEASE

A New and Serious Disease Recently Discovered in Pennsylvania[1]

C. R. Orton and F. D. Kern

Part I contains facts of general interest concerning this disease; Part II contains facts of a technical nature with regard to the life history and development of the parasite causing the disease concerning which there has been a rather widespread interest.

PART I

A most serious disease of the potato, which is well described by the common name, Potato Wart, has been found to be fairly well established in Luzerne County, Pennsylvania. This disease which has been also called Black Wart, Black Scab, Warty Disease, Cauliflower Disease and Potato Canker, has been known to science since 1896, when it was described in Hungary[2]. The first published reports of its presence in Great Britain appeared in 1902,[3] but there is evidence to show that it was present many years without being recognized. It was certainly there in 1899 and possibly twenty years before that. Since that time it has become widely distributed in districts in England, Wales, Scotland and Ireland. In 1909 the disease made its appearance on this side of the Atlantic Ocean, having been discovered in Newfoundland by the Dominion botanist[4].

On account of the fact that the disease was of such a nature that it might be easily introduced on imported tubers, and since importations were being made into the United States from both Europe and Newfoundland during the time the disease was spreading in those places, it was thought by many that this disease was liable to be introduced into this country. Being without a doubt one of the mose serious diseases of the potato, the possibility of its

1. Contribution from the Department of Botany, The Pennsylvania State College, No. 13.
2. Schilberszky, Ein neuer Schorfparasit der Kartoffelknollen. Ber.d. Deutsch. Bot. Gesell. 14: 36. 1896.
3. Carruthers, W., Diseases of Trees and Plants. The Jour. of the Royal Agri. Soc. of England, 63: 292. 1902.
   Potter, M. C. A new Potato Disease, The Jour. of the Board of Agriculture, 9: 320. 1902-3.
4. Gussow, H. T., A Serious Potato Disease Occurring in Newfoundland. Bull. No. 63, Department of Agriculture, Central Experiment Farm, Ottawa, Canada, October, 1909.

introduction was naturally viewed with considerable alarm. In March, 1910, W. A. Orton and Ethel C. Field, of the U. S. Department of Agriculture, issued a warning circular (No. 52) entitled "Wart Disease of the Potato; A Dangerous European Disease Liable to be Imported into the United States". Two years later Spaulding and Field in a Farmers' Bulletin (No. 489) of the U. S. Department of Agriculture discussed the possible entrance of potato wart under the caption "Two Dangerous Imported Plant Diseases". In the same year, 1912, Congress passed a bill known as "The plant quarantine act", establishing a Federal Horticultural Board with powers to regulate importation of plants and plant products for the purpose of preventing the introduction of diseases and pests. It is interesting to note that Potato Wart was considered so important that it was the basis of Quarantine No. 3. No. 1 was directed against White Pine Blister Rust, and No. 2 against The Mediterranean Fruit Fly. The act was approved August 20, 1912, and the notice of the quarantine against Potato Wart was issued just one month later. By this quarantine the importation of potatoes from the countries where Wart was then known to exist was prohibited, viz., Newfoundland, the Islands of St. Pierre and Miquelon, Great Britain, including England, Scotland, Wales and Ireland, Germany, and Austria-Hungary. This quarantine is still in effect.

As the importations ceased and the years passed by without the discovery of the deadly malady it seemed certain that the United States had avoided this trouble. Now the disease turns up most unexpectedly in east central Pennsylvania several years after the original anxiety concerning its introduction had ceased. The disease was first recognized by Mr. J. G. Sanders, Economic Zoologist of Pennsylvania, from diseased specimens of potatoes forwarded to him about the middle of September, 1918, from Highlands, Luzerne County. There is evidence that the disease has been present in this region for several years, but this is the first definite recognition and report of its presence in continental North America.

The infested district in Pennsylvania is a mining region, there being no land under cultivation except the gardens, the produce of which is grown for home consumption, and there are no contiguous cultivated areas. It does not seem likely that potatoes, garden truck, or other plants or plant products, which might carry the disease, will be sent out of the region, and yet the disease has been

so virulent and has spread so rapidly in other countries where it has become established, that it seems advisable to spread widely the news of the situation, so that all potato growers may know of the possible danger. Every potato grower in the state, and especially those in the region of the infested area, should be on the lookout for this disease. It is hoped that the disease may be checked before it spreads to other localities, but this can be effected only by timely effort and thorough co-operation. We would dislike to cause any unnecessary alarm among potato growers, but this disease is so disastrous, not only in entirely ruining a crop which it attacks, but in rendering the soil unfit for potato cultivation for several years, that we feel it necessary to send out this warning.

Symptoms of the Disease

In its usual development the disease is characterized by prominent wart-like outgrowth on the tubers. These warty excresences on the tubers are responsible for the common names "potato wart", "warty disease", "canker", and "cauliflower disease". The warty growths vary in size and distribution. For the most part they appear on the tubers, having their origin in the eyes (Fig. 1), but they may occur on the sprouts which develop new tubers, on the lower portions of the stem, and possibly on the roots. The warty masses are often suggestive of the crown gall disease known on various plants. Being entirely underground the disease is not evident until digging time, although some observers have pointed out a tendency for the tops of badly diseased plants to remain green longer than in the case of healthy or only slightly affected plants.

The warts are at first whitish or of the color of the young tubers, but eventually they turn to a darker shade, a fact which is responsible for the name "black wart" in some localities. When disturbed and left exposed on the ground, the warty masses may continue growth and often take on a green color. The warty tumors vary from the size of a pea to growths which may equal or exceed the size of the tuber from which they arise (Fig. 1). Where smaller growths occur the remainder of the tuber may appear quite normal. In severe cases the whole surface of the tuber may become involved, thus presenting a large warty mass which has lost all resemblance to a potato (See Frontispiece). In diseased hills some tubers may appear on cursory examination to be healthy, but the eyes are likely

to be infected. In the early stages, it may be difficult or even impossible to recognize the presence of the disease, and it is by the use of such apparently sound tubers for seed that the disease has been spread in the regions where it has been introduced. The photographs illustrate in a better way than words, the general appearance of the disease.

Fig. 1. Typical appearance of individual potato tubers infected with the potato wart organism. Note that the two upper middle tubers have been cut lengthwise to show the relationship between the warts and the "eyes". It is also to be noted that the vascular system extends into these warty growths. Tubers like the two lower, middle ones are easily overlooked in the bin or during digging.

Potato Wart Distribution in Pennsylvania

Immediately following the discovery of potato "wart" in the state, representatives of the Offices of Cotton, Truck and Forage Crop Disease Investigations, and Plant Disease Survey of the Bureau of Plant Industry, Washington, D. C., the Bureau of Economic Zoology, Harrisburg, Pa., and The Pennsylvania State College, assembled at Freeland and laid plans to survey the anthracite region in order to ascertain the present distribution of the disease. The survey work was pushed rapidly owing to the lateness of the season and the large amount of territory to be covered.

The anthracite coal fields are really divided into two regions; one extending from a few miles northeast of Freeland, Pa., southward to Mauch Chunk, and eastward to a few miles north of Harrisburg. The other region extends from Forest City along the eastern branch of the Susquehanna River to Shickshinny. In these two regions there are thousands of miners' gardens where the disease might be present. Eighteen men[5] took part in the survey, travelling by automobile, trolley and rail. In this way practically every city and village has been visited and thousands of gardens and bins inspected. At the present time the disease has been found in twenty-six towns and three counties, as follows: **Carbon County,** Coxville, Junedale (Leviston), Mauch Chunk; **Luzerne County,** Beaver Brook, Crystal Ridge, Cranberry, Drifton No. 1, Ebervale No. 1, Ebervale No. 2, Ebervale No. 3, Eckley, Freeland, Harwood, Hazleton, Hazelwood, Highlands, Humboldt, Jeanesville, Sandy Run, Slabtown, Stockton No. 6, Stockton No. 7, Swamptown, Upper Lehigh; **Schuylkill County,** Jackson, Sheppton.

Fortunately, the disease has not been found in the Susquehanna Valley of the anthracite region, for if it once becomes established there, the danger of its spread would be much greater, owing to the topography of that region which has a common drainage system where the spores of the fungus might be widely scattered to the trucking sections on the river bottom during heavy rains and spring freshets. However, potatoes are not grown in this section to the extent they are in the Hazleton district, which may account for the apparent absence of the disease.

Preventive Measures

For the prevention of any disease there are four lines of attack, namely, **exclusion, eradication, protection** and **immunization.** The attempt to **exclude** potato "wart" from the United States has failed, because the disease was, in all probability, introduced into our midst before the Federal quarantine was placed against it. It is possible, however, to exclude the disease from other localities in the state and from other states by placing a guard in the form of a strict quarantine

5. The following men took part in the preliminary survey for potato wart in Pennsylvania: Messrs. D. C. Babcock, M. L. Benn, A. W. Buckman, I. C. Cherrington, D. L. Dolbin, H. A. Edson, A. O. Finn, T. L. Guyton, L. O. Kunkle, J. M. LeCato, F. C. Meier, E. W. Olive, C. R. Orton, E. F. Pierce, J. G. Sanders, M. Shapovalov, H. S. Stahl, G. B. Stichter.

around the infested region. This will be done promptly by the Bureau of Economic Zoology of the State Department of Agriculture, which office by law controls the inspection and movement of nursery stocks and other plant materials. Any regulatory actions must receive the heartiest support from all concerned if this terrible disease is to be held in check.

No method of **eradicating** the disease has been attempted on a large enough scale to enable us to judge whether or not this can be done. As the parasite is known to live in the soil for several years, at least under some conditions, the process of starving it out is manifestly a slow one. Whether soil sterilization with steam or chemicals may be efficient remains to be proved.

There is no way known of **protecting** potato plants from infection by this fungus by spraying, seed treatment, and the like. Therefore, potatoes must **not** be grown in infested soil until such soil has been shown to be free of the parasite.

In the British Isles considerable advance has been made along the line of obtaining varieties of potatoes **immune** to potato "wart". Several varieties have been found which show a remarkable degree of resistance to this disease. It is our intention to secure the most promising of these varieties and test them out under our conditions along with the leading varieties already grown in this country. It is to be hoped that some variety will be found which is immune to this disease.

As the fungus no doubt survives the digestive and alimentary tracts of animals and may be spread in the manure, it is extremely important that any diseased tubers or peelings from such tubers, be boiled before feeding in order to kill the fungus. It is also quite apparent that the habit of throwing potato peelings on the garden for the poultry to pick up has been one of the commonest methods of spreading this disease in this state and is one which should be discontinued forthwith. If it is desired to feed such refuse it can be made safe by boiling. If not used for feeding purposes it should be completely destroyed by burning.

In view of the national and international importance of potato "wart" it is the duty of every person who grows potatoes, whether in the field or garden, to be extremely cautious to use for seed only tubers which are healthy in every detail of appearance. Slight in-

fections are easily overlooked and great care must be taken. Anyone observing suspicious cases should feel it their duty to report them at once, so that expert advice can be given. Only by such cooperative spirit can we hope successfully to combat and eradicate potato wart.

PART II.

Cause of the Disease

Potato "wart" is caused by a member of one of the lower orders of fungi, the Chytridiales. This organism was described under the name *Chrysophlyctis endobiotica* by Schilberszky in 1896. In 1910 Percival[6], an English botanist, published an account of this fungus, and decided that it belonged to another genus of this order. He, therefore, proposed the combination *Synchytrium endobioticum* (Schilb.) Percival.

Massee[7], another English botanist, who considered the potato "wart" disease as it occurred in the British Isles to be distinct from the disease described by Schilberszky in Hungary, gave the causal fungus a new name *Synchytrium Solani.* For the present, we shall use Schilberszky's name to designate the fungus causing potato "wart".

Life History of the Parasite

The published accounts of the life history of *Chrysophlyctis endobiotica* are more or less conflicting. It is certain that this fungus does not develop at any time a mycelium of thread-like elements, such as form the vegetative phase of most fungi. The fungus lives entirely within the host cells. An examination under the microscope of a bit of the diseased tissue taken in the autumn from one of the warts, reveals the fungus in the host cells just below the surface. In infected cells may be seen conspicuous rusty brown bodies which are the reproductive organs of the parasite. The brownish structures are globoid or oval in shape and when mature have a thick outer wall for the protection of the contents. The wall is rough with somewhat irregular ridges (Fig. 2).

---

6. Percival, John. Potato Wart Disease: the life history and cytology of Synchitrium endobioticum (Schilb.) Perc. Centralblatt f. Bakt. Paras. u. Infektionskr. **25**: 440-446. Taf. I, II, III. 1910.
7. Massee, George. Diseases of Cultivated Plants and Trees, 98-103 (Text book). 1913.

By the decomposition of the rather fragile host tissue comprising the warts, these bodies escape into the soil and it is in this way that the soil becomes infested. These heavy-walled bodies are a resting condition of the fungus tiding it over from one growing season to another. It is certain that they may remain alive for several

Fig. 2. Diagrammatic view of cross section of a mature sporangium containing spores which are apparently mature. The old host nucleus is clearly seen lying embedded in the spore mass and remaining cytoplasm. Its shriveled outline is evident. A delicate membrane encloses the whole spore mass inside of the thick rough wall. Magnified about 1400×.

years. It is reported on good authority that soil containing them may retain its infecting power for six to eight years. For a long time the form of activity which these resting bodies assumed when they produced new infections was obscure. All investigators now agree that they divide internally into numerous spores which finally possess the power of moving about, hence known as swarm-spores. There is a great deal of uncertainty as to the manner in which these gain entrance to the potato cells. We have had no opportunity to make any observations on this point. It is certain that these swarm-spores are the simplest stage in the life-history of the fungus, and that they are the actual infecting agents. This makes it evident that the heavy-walled bodies are spore-cases, or sporangia. Just what conditions are necessary for the formation and liberation of the swarm-spores from these sporangia is also unknown. It seems probable that not only suitable soil conditions as regards moisture and temperature are required, but, perhaps, also the proximity of living potato plants.

After entry into the host the swarm-spores of the parasite become imbedded in the living cytoplasm of the host cells. Several

uninucleated bodies which are aparently swarm spores may be observed in a single host-cell, frequently as many as twelve or more having been observed. They show a very marked tendency to

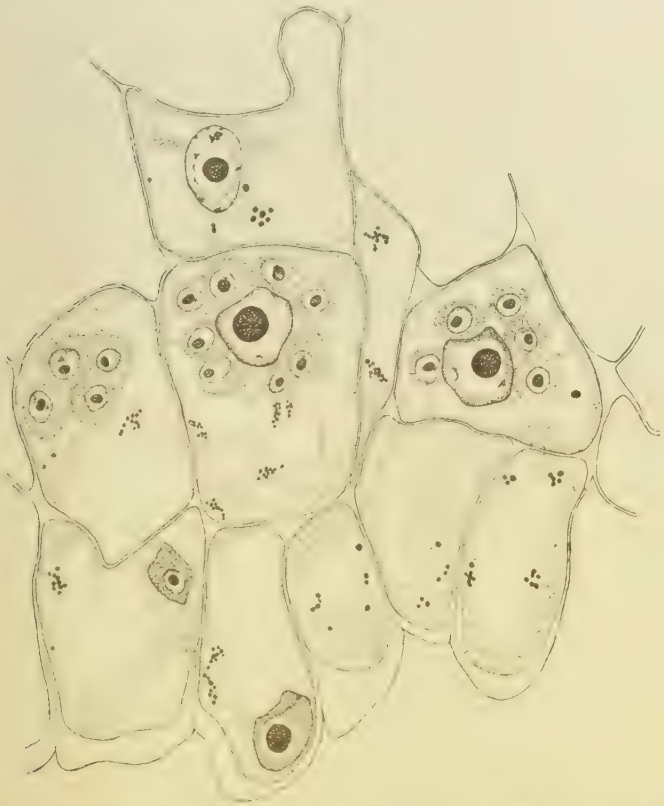


Fig. 3. A section through the external tissue of the warty growth. The cells below are the outermost layer. Host nuclei are shown in four of the cells. In two of these the parasite is shown in the form of plasmodia-like bodies surrounding the host nucleus. The small, dark bodies are the centers around which starch grains develop. Magnified about 1400×.

cluster around the nucleus of the host cell (Fig. 3). Whether these parasitic bodies may migrate from one host cell to another is not clear from our observations. It seems certain that in infected cells containing several of the above mentioned bodies they coalesce in

some way to form a single mass, although good series showing fusions have not been found. The fusions are, apparently, of the nature of cell-fusions in which the nuclei are not involved. The fungus, which is now in the form of a plasmodium, increases rapidly in size until it may nearly fill the host cell and becomes surrounded with a rather thin, but distinct wall. In any microscopic preparation in which these points are being observed there may be seen in the developing sporangium within the infected cells a distinct nucleus of large size, frequently one-fourth to one-third the diameter of the whole mass and containing a prominent nucleolus.

The cytoplasm of the fungus shows a reticulate condition, with numerous prominent denser bodies. The large nucleus, usually centrally placed, presents a very striking appearance, and it is little wonder that its significance and behavior should be the subject of considerable interest. Percival[8], who investigated Potato Wart several years ago, assumed without question that this large nucleus was the primary nucleus of the developing fungous body. He did not discuss its origin, but stated that it is at first small, increasing at a rapid rate until it is 16-18 microns in diameter. It was never seen dividing mitotically, but Percival describes its gradual disintegration by the transfer of its chromatin in the form of granules or globules to the meshes of the fungous cytoplasm. These chromatin granules are by him held responsible for the organization of the nuclei of the new swarm-spores. He also describes the resultant shrinkage of the primary nucleus.

Our investigations lead us to an entirely different interpretation of this part of the life-history. We believe this large nucleus to be the host-cell nucleus which has been completely surrounded by the fungus. This view is suggested by the fact that various stages in the process of engulfment have been found (Fig. 4), and is supported on the one hand by the fact that it agrees with the nuclei of normal host cells in structure and size, and on the other hand by its failure to take any part in the spore production of the fungus. It appears among the spores as a shrivelled, dark body in which the nucleole may still be seen. The indistinct, denser bodies of the reticulated fungous cytoplasm are the fungous nuclei which have arisen by multiplication of those contributed by the infecting swarm-spores. Divisions have continued until the final nuclei of the re-

8. In the paper previously referred to under the section "Cause of the Disease".

sultant spores, into which the reticulated mass ultimately divide, are very small. The process of spore formation is not clear. During the process of spore production the wall of the sporangium gradually increases in thickness and when mature is marked with irregular ridges. In the immature stages infoldings of the thickening wall are believed to be the beginnings of the surface sculpturing (Fig. 4). The production of these thick-walled "resting" sporangia completes the story of the life-cycle to the point where this account begins.

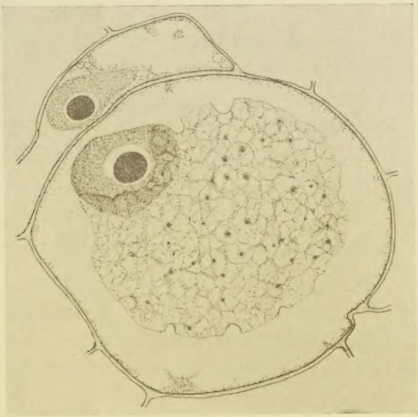


Fig. 4. An infected potato cell showing the developing parasite with its sculptured outer membrane and its numerous small nuclei; the large host nucleus and the remains of the host cytoplasm just inside the wall. The adjoining cell is partially collapsed but clearly shows the relative size and structure of its nucleus when compared with the nucleus of the parasitized cell. The fungous mass is very evidently in the process of enveloping the host nucleus. Magnified about 1400×.

It should be said at this point that Percival mentions thin-walled sporangia which are produced in small groups during the summer. We have not observed anything corresponding to this description, but we have not worked with material collected during the summer. These summer sporangia are reported to differ chiefly in the thinner walls. The large nucleus is described as a feature of the development. In our material have been observed some fungous bodies in which the cytoplasm appears homogeneous and granular rather than reticulated. They are enclosed in a thin, even wall,

and finally possess numerous extremely small nuclei. These have not been found to contain the host nucleus. Transitions from these bodies into thick-walled sporangia have not been seen. Whether they bear any relationship to the thin-walled sporangia described by Percival, or what part they may play in the life-history of the parasite is not now clear.

Anatomy of the Diseased Tissues

The "warts" produced by the parasite are most interesting structures. They are more or less spongy in texture and are easily broken up and separated from the parent tissue. On the tubers they seem always to arise from the eyes; the reason for this probably being that the eye is a bud and is the only place where formative tissue is exposed to infection.

A longitudinal section through these warts shows this growth to be made up of two sorts of tissue—fundamental and vascular. The fundamental tissue appears about normal, except at or near the surface of the warts. At these points the cells are smaller and appear to be more richly supplied with cytoplasm. Starch is found in abundance in the deeper cells of this tissue.

The vascular tissue presents more abnormal conditions. In the deeper portions of the warts water conducting cells are found. These conducting cells with spiral thickening, rarely extend to the region near the surface of the warts. Thin-walled, elongated cells, rich in cytoplasm, similar in appearance to procambial strands are very abundant and these strands branch and ramify through the external parts of the warts to their very surface. Apparently in this diseased vascular tissue the water conducting cells are later differentiated.

The vascular strands contain cells which appear to be capable of division at any point. Division figures have been seen deep in the tissue as well as near the surface. This fact apparently explains the irregular surface of the warts, for where the strands extend to the surface those regions grow most rapidly.

The surface of the warty growths is covered by a single layer of differentiated cells which are very similar to the surface cells of a growing point. This layer of cells is not covered with a protective cuticle and the individual cells remain in an active state cap-

able of division so long as the warts continue to grow. As soon as growth ceases disintegration takes place rapidly, and the whole tissue decays and eventually disappears.

The effect of the parasite upon the tissue is stimulative to rapid cell division, but only rarely to the enlargement of individual cells. The growth is, therefore, a hyperplasia rather than a hypertrophy.

The mature sporangia nearly fill the cells they inhabit, with the result that the surrounding host cells are frequently distorted as a result of the pressure placed upon them. This often produces an effect which appears as though the parasitized cells were enlarged, but this is more apparent than real, when careful comparisons are made.

The parasite is confined almost entirely to the outer five or six layers of cells. Only occasionally are sporangia seen in the deeper tissues and in no cases have swarm-spores been found in any but the outer layers.

The most interesting phenomenon exhibited by this parasite is its relationship to the host nucleus of the infected cells. There seems to be first of all an attraction between the swarm cells of the parasite and the nucleus of the host. The nature of this attraction we shall not attempt to discuss here. It is evident, however, that these swarm-cells which are not bounded by a cell wall, mass together about the host nucleus (Fig. 3), and soon a fusion of their cytoplasm results. It is not until after this cell fusion that a wall is developed and this wall is at first quite indefinite. It appears from our preparations that the host nucleus in a majority of cases is included within the developing sporangium at the time of the fusion of the swarm-spores. In other cases the host nucleus does not become enclosed within the parasite during fusion of the swarm-cells. In such cases we have seen numerous instances where the fused mass of the parasite apparently engulfs the host nucleus later. This occurs before the membrane becomes thickened around the developing sporangium. The process of engulfment appears to be one of invagination (Fig. 4). At first the membrane gradually enfolds the host nucleus until it is entirely enveloped; then apparently the enveloping edges of the membrane fuse and the host nucleus moves to the center of the sporangium and further development proceeds normally.

After the host nucleus comes to lie in the center of the developing sporangium, certain changes in its appearance are then noted. It becomes spherical, probably as the result of the equal pressure from all sides and also, perhaps, from its greater sap pressure. There then appears a reorganization of its contents. Its cytoplasmic contents become less homogeneous. The chromatin is aggregated in more or less dense masses near the nucleole, the vacuole about the nucleole disappears and the less dense cytoplasm is seen collected close to the nuclear wall. It appears that the chromatin undergoes changes in position and form not greatly different from the changes which take place in the normal nucleus previous to division. It, however, never divides so far as we have observed, nor do we find any reference in the literature to the division of the nucleus. The sporangium then passes through its developmental stages, during which the host nucleus retains its size and appearance until spore formation, when it loses its turgidity, its wall collapses, and it is then seen as a denser staining mass among the spores in the center of the sporangium (Fig. 2).

Such a case of parasitism is apparently new among plants. In the Erysiphaceae it has been noted by Harper[9], that the haustoria are enveloped by a sheath which is apparently the remains of the host nucleus. Rosen[10] observed in *Puccinia asarina* that the haustoria were generally in close relation with the host nuclei, and Ward[11] observed a similar phenomenon in *Puccinia dispersa*. Colley[12] and Dodge and Adams[13] have observed other cases of this sort between haustoria and nuclei of host cells in species of *Peridermium*.

In view of the fact that the parasite dealt with in this bulletin apparently does not always engulf the host nucleus the significance of this act is not clear. The further development of sporangia which do not contain the host nucleus must be followed out before it becomes evident what effect the host nucleus has upon the parasite.

---

9. Harper, R. A., Ueber das Verhalten der Kerne bei der Fruchtentwickelung einiger Ascomyceten. Jahr. Wiss. Bot. 29: 664. 1896.
10. Rosen, F., Betrage zur Kenntniss der Pflanzenzellen. Cohn's Beitr. Biol. Pflanzen 6: 258. 1893.
11. Ward, H. Marshall. Phil. Trans. Royal Soc. 196: 29. 1903.
12. Colley, R. H., Diagnosing White Pine Blister Rust from its Mycelium. Jour. Agr. Research 11: 284. 1917.
13. Dodge, B. O., and Adams, J. F., Observations on the development of Peridermium cerebrum. Mem. Torrey Club 17: 256-257. 1918.